EFFECT OF CORIOLIS ACCELERATION ACCUMULATION ON CATECHOLAMINE EXCRETION

N. S. Nemchenko

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EFFECT OF CORIOLIS ACCELERATION ACCUMULATION ON CATECHOLAMINE EXCRETION

N. S. Nemchenko

The purpose of the present project was to study the physiological level of catecholamine excretion in healthy persons and to determine the functional state of the sympathoadrenal system as a function of the degree of sensitivity to motion sickness.

The motion sickness was caused by Coriolis acceleration accumulation (by the method developed by A. Ye. Kurashvili, 1967). The rotation program called for angular acceleration of 22.5 degrees/sec² for a period of 4 sec, followed by a uniform speed of 90 degrees/sec (the duration of the effect being about 7 min). The subject, with his eyes closed, was rotated in a chair actuated by an electric motor from a control panel. After the sensation of rotation had vanished, the subject began (on command) to tilt his head in the direction of the left shoulder and then in the direction of the right, with the chair continuing to rotate. Tilting and straightening of the head were performed every 10 sec (a total of 5 times). After a 10 second pause (if the subject tolerated the motion effect well), the experiment was repeated with the chair rotated in the opposite direction. The intensity of vestibular reactions was determined on the basis of pulse and respiration rate, body temperature, amount of perspiration, and the subjective sensations of the subjects. The catecholamines were determined in the 24 hour urine by the fluorometric method of Euler and Lishako as

We applied the method indicated above for study of 90 persons health to all intensive purposes ranging in age from 18 to 42. They were all divided into three groups (according to the classification of K. L. Khilov, 1952) on the basis of the intensity of vegetative reactions and subjective sensations in

modified by E. M. Matlina (1961) before and after application of the motion

effect.

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^{*}Numbers in the margin indicate foreign pagination.

response to stimulation of the vestibular apparatus by Coriolis acceleration accumulation. The first group (17 persons) included persons insensitive to motion sickness, ones from whom subjective or objective symptoms of such sickness were absent. First-stage vestibulovegetative reactions were observed in the subjects of the second group (29), and second-stage and third-stage reactions in those of the third group (44).

The average adrenalin content before application of the motion effect was 6.24 µg/24 hours in the subjects of the first group, 10.46 in those of the second, and 7.25 in those of the third. After the application of the acceleration effect adrenaline excretion in the persons of the first group increased to 11.11 μ g/24 hours (P < 0.01), while it decreased (6.55) in those of the second group. It decreased (4.86) in one-half of the subjects of the third group and increased in those of the other half.

Determination of the noradrenalin excretion level revealed that the highest /56 content in the urine prior to application of the motion effect were typical of persons sensitive to motion sickness. Increase in the noradrenalin content (on the average 22.54 $\mu g/24$ hours) in comparison to the initial level (16.61), on the other hand, was observed in response to the acceleration in the subjects of the first group. The difference in average values is reliable (0.01 < P << 0.05). In the persons sensitive to the motion effect, however, there was observed a decrease in noradrenalin secretion, which was the most pronounced in the case of second-stage vestibulovegetative instability (third group). Thus, while the noradrenalin excretion was 27.23 µg/24 hours prior to application of the motion effect, it was 14.36 after such application (0.01 < P < 0.05).

We know that adrenalin intensifies the transmission of nerve pulses in the sympathetic ganglia, while noradrenalin inhibits such transmission in the parasympathetic ganglia. The statistically reliable increase in adrenalin and noradrenalin excretion in our studies in response to stimulation of the vestibular apparatus by Coriolis acceleration accumulation in persons insensitive to motion sickness apparently indicates good tonus both of the hormonal and of the mediator elements of the sympathoadrenal system. The decrease in the adrenalin content, and in some instances in that of adrenalin as well, in persons of the second group indicates suppression of the sympathetic division of the vegetative

nervous system and predominance of the tonus of the parasympathetic division, this creating a predisposition toward development of the motion sickness syndrome. The statistically reliable decrease in noradrenalin excretion, and in many instances also the increase in adrenalin, in response to application of the motion effect in subjects of the third group in our opinion reflects sudden stress on the sympathoadrenal system, a stress manifested in hormonal-mediator dissociation and in reduction of the activity of the mediator component of this system.

In order to verify our assumptions we subjected 30 persons of varying degrees of vestibulovegetative instability to repeated application of the motion effect (by the method described above), one hour after they had taken 20 drops of a 0.1% solution of adrenalin in 25-30 ml of water on an empty stomach. Analysis of the physiological indices and subjective sensations of these persons showed that in 10 of the 13 persons who had previously exhibited first-stage motion sickness and in 8 of the 17 exhibiting the second and third stages, the vestibulovegetative reactions were less pronounced when they were again subjected to the action of accumulated Coriolis acceleration. The intensity of the reactions remained the same in the other subjects.

Analysis of the average values of catecholamine excretion in response to repeated subjection to the action of accumulated Coriolis accelerations after the taking of adrenalin permits the conclusion that increase in the excretion both of noradrenalin (22.61 $\mu g/24$ hours prior to application of the motion effect and 31.38 after the application) and of adrenalin (2.76 and 6.98 respectively) is found in persons exhibiting less pronounced vestibulovegetative reactions. The difference in the average values is statistically reliable. In the subjects in whom the adrenalin exerted no positive effect the increase in elimination of catecholamines with the urine was insignificant.

Thus a definite relationship exists between the degree of sensitivity to motion sickness and the catecholamine excretion level. Moderate increase in the excretion both of adrenalin and of noradrenalin is found in persons insensitive to motion sickness in response to accumulated Coriolis acceleration. In persons sensitive to motion sickness, on the other hand, there is observed a tendency toward decrease in the noradrenalin concentration, a decrease which is the most

pronounced in the case of second-stage vestibulovegetative instability. Adrenatin administered orally prior to application of the motion effect increases the resistance to its action chiefly in persons in whom first-stage vestibulovegetative reactions developed on stimulation of the vestibular apparatus by accumulated Coriolis accelerations.

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